

. . . . .  
 . . . . .

Fasting Blood Glucose has the Best Correlation with HbA<sub>1c</sub> in Type 2 Diabetes

Kim HJ, Lee TH, Choi SH, Kim SK, Kim DJ, Rhee YM, Kim SH  
 Ahn CW, Cha BS, Song YD, Lim SK, Kim KR, Lee HC, and Huh KB

*Department of Internal Medicine Yonsei University College of Medicine, Seoul, Korea*

ABSTRACT

**Background:** We have designed a study to evaluate the relative value of Capillary blood glucose measurement at different times of the day in comparison with HbA<sub>1c</sub> measurement in assessing the mean glycemic control of type 2 diabetic patients.

**Methods:** The 118 patients with type 2 diabetes, all of whom very regular visitors of the outpatient clinic of the Diabetic Center of the Yonsei University Hospital, were entered consecutively into the study. Three months before enrollment, the dietary habits of each participant were checked, and the patients received recommendations for continuing their usual diet throughout the entire period of the study. The first sample was examined before breakfast at 6:00 A.M. (prebreakfast glucose and HbA<sub>1c</sub>); the second, at noon (prelunch glucose); and the third, at 5:00 P.M. (presupper glucose). The last sample was examined at 10:00 P.M. (bedtime glucose). The capillary whole blood glucose was used to determine blood glucose concentrations (by Surestep). A high-pressure liquid chromatography assay (VARIANT , BIO-RAD) was used to make all determinations of HbA<sub>1c</sub> (normal range, 4~6%). Simple linear regression analysis was used to examine the relationship between capillary blood glucose and HbA<sub>1c</sub> at each time point. Multiple

linear regression analysis was performed to determine which of the values of various capillary blood glucoses were significant and independent predictors of HbA1c.

**Results:** Mean concentration of capillary blood glucose at 6 A.M. is significantly different between three groups classified by levels of HbA1c, but not at other times. Simple linear regression analysis demonstrated that R-squares between the blood glucose and HbA1c were 0.187 (6:00 A.M.), 0.035 (noon), 0.116 (5:00 P.M.), and 0.108 (10:00 P.M.). Levels of the blood glucose at 6:00 A.M.; 5:00 P.M.; and 10:00 P.M. were correlated with HbA1c, but that at noon was not. Multiple regression analysis demonstrated that the standardized coefficients ( $\beta$ ) of the glucose levels at 6:00 A.M.; noon; 5:00 P.M.; and 10:00 P.M. were 0.348 ( $P<0.05$ ), -0.091 ( $P=0.369$ ), 0.168 ( $P=0.148$ ), and 0.103 ( $P=0.338$ ), respectively. Only, the blood glucose at 6:00 A.M. influenced to HbA1c.

**Conclusions:** In type 2 diabetes, the blood glucose at 6:00 A.M. have most strong correlation with HbA1c, and is better predictor of glycemic control than those of other times.

**Key Words:** Fasting blood glucose, HbA1c, Type 2 diabetes, Glycemic control

---

---

---

가

가

1-2),

가 .

3-4)

가

5-6)

가

가

8)

가

7)

가

4

가

2 118  
 16 82 ( , 56±13 )  
 17 42 kg/m<sup>2</sup> ( , 22.59±  
 4.25 kg/m<sup>2</sup>)  
 98  
 20  
 3

가

7 8  
 1  
 6 7  
 6  
 5  
 10

Surestep  
 high  
 pressure liquid chromatography assay (VARIANT  
 , BIO-RAD)

8.0% , 8.0%  
 10.0% , 10.0%

SPSS10.0

118

(HbA<sub>1c</sub><8%, n=16),  
 (8%HbA<sub>1c</sub><10%, n=35),  
 (10%HbA<sub>1c</sub>, n=67)

6

( vs

; p=0.008,

vs

; p<0.001,

vs

; p=0.026).

(p=0.047).

5

10

(p<0.05)

(Table 1).

6

가

10

가

5

Table 1. Mean Blood Glucose Concentrations (mg/dL) with Glycemic Control (Comparisons by One-way ANOVA)

Time	Total (n = 118)	Good control HbA <sub>1c</sub> < 8% (n = 16)	Moderate control 8% HbA <sub>1c</sub> < 10% (n = 35)	Poor control 10% HbA <sub>1c</sub> (n = 67)
6 A.M. CBG (mmol/L)	10.8 ± 3.9*	7.0 ± 2.1*†‡	10.2 ± 3.5†	12.1 ± 3.8*
Noon CBG (mmol/L)	11.9 ± 4.7*	9.6 ± 4.1†	11.3 ± 4.3	12.7 ± 4.8*
5 P.M. CBG (mmol/L)	11.7 ± 4.6*	8.3 ± 2.8†	10.4 ± 3.9†	13.2 ± 4.7
10 P.M. CBG (mmol/L)	13.6 ± 4.9	10.6 ± 3.8†	12.1 ± 5.1†	15.1 ± 4.5

\*; Significantly different from 10 P.M. capillary blood glucose

†; Significantly different from group with poor control of HbA<sub>1c</sub>

‡; Significantly different from group with moderate control of HbA<sub>1c</sub>

CBG; capillary blood glucose

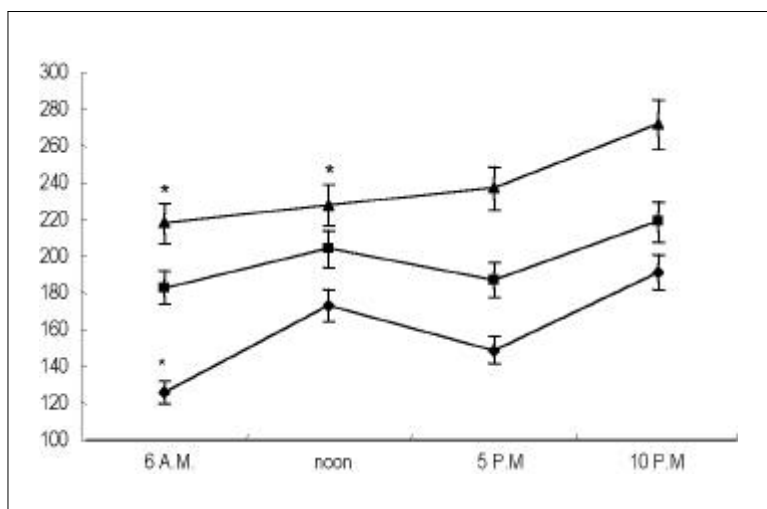


Fig. 1. Glycemic profile according to the level of glycemic control.

■, good control; ◆, moderate control; ▲, poor control.

\*; Significantly different from 10 P.M. capillary blood glucose

(R-square) 0.19 (p<0.001), 0.04

5 (p=0.43), 0.12 (p<0.001), 0.11 (p<0.001)

(Fig. 1).

10

(Table 2).

Table 2. Correlation Between Blood Glucose and HbA<sub>1c</sub>

Time	CBG	R-square	P value
6 A.M.	10.8 ± 3.9	0.19	<0.001
Noon	11.9 ± 4.7	0.04	0.43
5 P.M.	11.7 ± 4.6	0.12	<0.001
10 P.M.	13.6 ± 4.9	0.11	<0.001

CBG; capillary blood glucose

Table 3. Result of Multiple Regression Analysis with HbA<sub>1c</sub> and Levels of Capillary Blood Glucos

	Beta	p-value
6 A.M. glucose	3.51	0.001
Noon glucose	-0.91	0.369
5 P.M. glucose	1.46	0.148
10 P.M. glucose	0.96	0.338

, 10 가 0.348 )  
 (p<0.05), -0.091 (p=0.369), 0.168 (p=0.148), 0.103 가  
 (p=0.338) 6

(Table 3).

가

가

10-11)

가

가

가

1993 Diabetes Control and Complication Trial

1

United Kingdom Prospective

Diabetes Study Group

2

. Graf

1978

2

가



12)

가 가 가

13-14)

. Pecoraro

가

1986

2

1

2

15)

16-17) . Avignon

1997

2

가

가

가

가

가

2

18) . de Veciana

21-22)

19)

가

가

가

23-24)

가

가

(83%:17%)

가

가

20)

• 2

가

•

가 <sup>25-27)</sup> Schmitz 19

가

가

2

4

24

<sup>28)</sup>

:

1

2

가

2

118

,

.

3

. 6

,

,

5

,

,

10

Surestep

high pressure

2

liquid chromatography assay (VARIANT , BIO-RAD)

가

,

,

가

6

2

가

가

,

6

,

,

5

가

,

10

(R-

square)가 0.187 (p<0.001), 0.035 (p=0.43), 0.116 (p<0.001), 0.108 (p<0.001)

6

,

5

,

10

6

: 2

,

,

5

,

10

가

가

가

0.348 (p<0.05), -0.091 (p=0.369),

0.168 (p=0.148), 0.103 (p=0.338)

6

가

- 
1. The Diabetes Control and Complications Trial Research Group: The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* 329:977-986, 1993
  2. Ohkubo Y, Kishikawa H, Araki E, Miyata T, Isami S, Motoyoshi S, Kojima Y, Furuyoshi N, Shichiri M: Intensive insulin therapy prevents the progression of diabetic microvascular complications in Japanese patients with non-insulin-dependent diabetes mellitus: a randomized prospective 6-year study. *Diabetes Res Clin Pract* 28:103-117, 1995
  3. Klein R: Hyperglycemia and microvascular and macrovascular disease in diabetes. *Diabetes Care* 18:258-268, 1995
  4. Kuusisto J, Mykkanen L, Pyorala K, Laasko M: NIDDM and its metabolic control predict coronary heart disease in elderly subjects. *Diabetes* 43:960-967, 1994

5. Boden G, Master RW, Gordon SS, Shuman CR, Owen OE: Monitoring metabolic control in diabetic outpatients with glycosylated hemoglobin. *Ann Intern Med* 92:357-360, 1980
6. Nathan DM, Singer DE, Hurxthal K, Goodson J: The clinical information value of the glycosylated hemoglobin assay. *N Engl J Med* 310:341-346, 1984
7. American Diabetes Association: Clinical Practice Guidelines 2000. *Diabetic Care* 23(Suppl.):S1, 2000
8. U.K. Prospective Diabetes Study (UKPDS) Group: Intensive blood-glucose control with sulfonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS33). *Lancet* 352:837-853, 1998
9. Jarrett RJ: The cardiovascular risk associated with impaired glucose tolerance. *Diabet Med* 13(Suppl. 2):S15-S19, 1996
10. de Vegt F, Dekker JM, Ruhe HG, Stehouwer CD, Nijpels G, Bouter LM, Heine RJ: Hyperglycemia is associated with all-cause and cardiovascular mortality in the Hoorn population: the Hoorn Study. *Diabetologia* 42:926-931, 1999
11. Graf RJ, Halter JB, Probe D Jr: Glycosylated hemoglobin in normal subjects and subjects with maturity-onset diabetes: evidence for a saturable system in man. *Diabetes* 27:834-839, 1978
12. el-Kebbi IM, Ziemer DC, Gallina DL, Phillips LS: Diabetes in Urban African-Americans. VI. Utility of Fasting or Random Glucose in Identifying Poor Glycemic Control. *Diabetes Care*



- 21:501-505, 1998
13. Glycemic Control in Non-Insulin-Using Patients with Type 2 Diabetes? *Diabetic Care* 22:904-907, 1999
  14. Pecoraro RE, Koepsell TD, Chen MS, Lipsky BA, Belcher DW, Inui TS: Comparative clinical reliability of fasting plasma glucose and glycosylated hemoglobin in non-insulin dependent diabetes mellitus. *Diabetes Care* 9:370-375, 1986
  15. Colwell JA: The feasibility of intensive insulin management in non-insulin-dependent diabetes mellitus: implications of the Veterans Affairs Cooperative Study on Glycemic Control and Complications in NIDDM. *Ann Intern Med* 124:131-135, 1996
  16. Turner R, Cull C, Holman R, for the United Kingdom Prospective Study Group: United Kingdom Prospective Study 17: a 9 year update of a randomized, controlled trial on the effect of improved metabolic control on complications in non-insulin-dependent diabetes mellitus. *Ann Intern Med* 124:136-145, 1996
  17. Avignon A, Radauceanu A, Monnier L: Non-fasting plasma glucose is a better marker of diabetic control than fasting plasma glucose in type 2 diabetes. *Diabetes Care* 20:1822-1826, 1997
  18. de Veciana M, Major CA, Morgan MA, Asrat T, Toohey JS, Lien JM, Evans AT: Postprandial versus preprandial blood glucose monitoring in women with gestational diabetes mellitus requiring insulin therapy. *N Engl J Med* 333:1237-1241, 1995
  19. American Diabetes Association. Postprandial Blood Glucose. *Diabetes Care* 24:775-778, 2001
  20. Huh KB: The role of insulin resistance in Korean patients with metabolic and cardiovascular disease. *Experta Medica* p7, 1993
  21. : .  
21(S1):S7-S14, 1997
  22. Dinneen S, Gerich J, Rizza R: Carbohydrate metabolism in non-insulin-dependent diabetes mellitus. *N Engl J Med* 327:707-713, 1992
  23. Bogardus C, Lillioja S, Howard BV, Reaven G, Mott D: Relationships between insulin secretion, insulin action, and FPG concentration in non-diabetic and non-insulin dependent diabetic subjects. *J Clin Invest* 74:1238-1246, 1984
  24. Haller H: Postprandial glucose and vascular disease. *Diabet Med* 14(Suppl. 3):S50-S56, 1997
  25. Lefebvre PJ, Scheen AJ: The postprandial state and risk of cardiovascular disease. *Diabet Med* 15(Suppl. 4):S63-S68, 1998
  26. Ceriello A: Acute hyperglycemia and oxidative stress generation. *Diabet Med* 14(Suppl. 3): S45-S49, 1997
  27. Ole Schmitz et al. HbA1c Does Not Reflect Prandial Plasma Glucose Excursions in Type 2 Diabetes. *Diabetes Care* 23:1859-1860, 2000